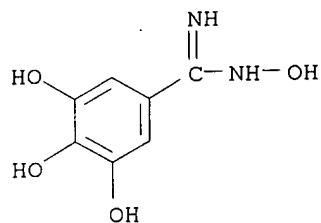
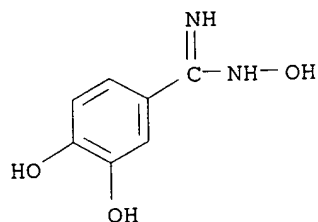


L2 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2000 ACS
RN 95933-74-7 REGISTRY
CN Benzenecarboximidamide, N,3,4,5-tetrahydroxy- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN N,3,4,5-Tetrahydroxybenzimidamide
CN **Trimidox**
CN VF 233
FS 3D CONCORD
MF C7 H8 N2 O4
CI COM
LC STN Files: ADISINSIGHT, BIOBUSINESS, BIOSIS, CA, CAPLUS, DRUGUPDATES,
IPA, PHAR, PROMT, TOXLINE, TOXLIT, USPATFULL



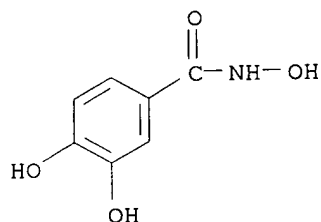
23 REFERENCES IN FILE CA (1967 TO DATE)
23 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L6 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2000 ACS
RN 95933-72-5 REGISTRY
CN Benzenecarboximidamide, N,3,4-trihydroxy- (9CI) (CA INDEX NAME)
OTHER NAMES:
CN **Amidox**
CN VF 236
FS 3D CONCORD
DR 125199-74-8
MF C7 H8 N2 O3
CI COM
LC STN Files: ADISINSIGHT, ANABSTR, BIOBUSINESS, BIOSIS, BIOTECHNO, CA,
CANCERLIT, CAPLUS, DDFU, DRUGPAT, DRUGU, DRUGUPDATES, EMBASE, IPA,
MEDLINE, PHAR, TOXLINE, TOXLIT, USPATFULL



15 REFERENCES IN FILE CA (1967 TO DATE)
1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
15 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L3 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2000 ACS
 RN 69839-83-4 REGISTRY
 CN Benzamide, N,3,4-trihydroxy- (9CI) (CA INDEX NAME)
 OTHER NAMES:
 CN 3,4-Dihydroxybenzohydroxamic acid
 CN 3,4-Dihydroxyphenylhydroxamic acid
 CN **Didox**
 CN N,3,4-Trihydroxybenzamide
 CN NSC 324360
 CN VF 147
 FS 3D CONCORD
 DR 106573-41-5
 MF C7 H7 N O4
 CI COM
 LC STN Files: ADISINSIGHT, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS,
 BIOTECHNO, CA, CANCERLIT, CAPLUS, CIN, DDFU, DRUGNL, DRUGU,
 DRUGUPDATES,
 EMBASE, IPA, MEDLINE, PHAR, PROMT, RTECS*, TOXLINE, TOXLIT, USPATFULL
 (*File contains numerically searchable property data)



51 REFERENCES IN FILE CA (1967 TO DATE)
 1 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
 51 REFERENCES IN FILE CAPLUS (1967 TO DATE)

L6 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2000 ACS
 AN 1999:228730 HCAPLUS
 DN 131:57143
 TI Nitric Oxide Up-regulates the Expression of Intercellular Adhesion
 Molecule-1 on Cancer Cells
 AU Toyoshima, Takahiko; Kamijo, Ryutaro; Takizawa, Kunio; Sumitani, Kaname;
 Hatori, Masashi; Nagumo, Masao
 CS Second Department of Oral and Maxillofacial Surgery, School of Dentistry,
 Showa University, Tokyo, 145-8515, Japan
 SO Biochem. Biophys. Res. Commun. (1999), 257(2), 395-399
 CODEN: BBRC9; ISSN: 0006-291X
 PB Academic Press
 DT Journal
 LA English
 CC 14-1 (Mammalian Pathological Biochemistry)
 Section cross-reference(s): 2, 3
 AB Nitric oxide (NO) is an unstable **free radical** that
 functions as a cytotoxic agent secreted by macrophages to kill
cancer cells. Here we report the effect of NO on the expression
 of intercellular adhesion mol.-1 (ICAM-1) on **cancer** cells. NO
 donors such as SNP, SNAP and SIN-1 up-regulated the expression of ICAM-1
 on NA cells, a squamous cell carcinoma cell line. Northern blot anal.
 showed that the induction of ICAM-1 might be due to transcriptional
 induction of ICAM-1 mRNA. Up-regulation of ICAM-1 mRNA by NO donors was
inhibited by carboxy-PTIO, a NO scavenger. Although **NF**
-kappa.B activity was induced by NO donors, AP-1 was
 not induced by them. Staurosporin, a protein kinase C (PKC)
inhibitor, **inhibited** the induction of ICAM-1 on NA cells
 by NO, whereas genistein, a protein tyrosine kinase **inhibitor**,
 did not. These findings indicate that NO up-regulates ICAM-1 expression
 on **cancer** cells by a regulatory mechanism involving PKC and
 suggest that **NF-kappa.B**, but not AP-1,
 might be involved in induction of ICAM-1 by NO in **cancer** cells.
 (c) 1999 Academic Press.
 ST nitric oxide ICAM1 squamous cell carcinoma NFkappaB
 IT Cell adhesion molecules
 RL: BOC (Biological occurrence); MFM (Metabolic formation); BIOL
 (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence)
 (ICAM-1 (intercellular adhesion mol. 1); nitric oxide regulation of
 ICAM-1 expression in human tongue squamous cell carcinoma cells)
 IT Transcription factors
 RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
 (NF-kappa.B (nuclear factor .kappa.B); nitric oxide regulation of
 ICAM-1 expression in human tongue squamous cell carcinoma cells)
 IT Tongue
 (squamous cell carcinoma; nitric oxide regulation of ICAM-1 expression
 in human tongue squamous cell carcinoma cells)
 IT 10102-43-9, Nitric oxide, biological studies
 RL: BAC (Biological activity or effector, except adverse); BIOL
 (Biological study)
 (nitric oxide regulation of ICAM-1 expression in human tongue squamous
 cell carcinoma cells)
 IT 141436-78-4, Protein kinase C
 RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
 (nitric oxide regulation of ICAM-1 expression in human tongue squamous
 cell carcinoma cells)
 RE.CNT 24
 RE

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L6 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2000 ACS

AN 1998:441049 HCAPLUS

DN 129:183931

TI In vivo inhibition of nitric oxide synthase gene expression by curcumin,
a

cancer preventive natural product with anti-inflammatory properties
AU Chan, Marion Man-Ying; Huang, Hsing-I.; Fenton, Marilyn Ruth; Fong, Dunne
CS Department of Biomedical Sciences, Pennsylvania College of Podiatric
Medicine, Philadelphia, PA, 19107, USA

SO Biochem. Pharmacol. (1998), 55(12), 1955-1962

CODEN: BCPCA6; ISSN: 0006-2952

PB Elsevier Science Inc.

DT Journal

LA English

CC 1-6 (Pharmacology)

Section cross-reference(s): 18

AB Curcumin is a naturally occurring, dietary polyphenolic phytochem. that
is

under preclin. trial evaluation for **cancer** preventive drug
development and whose working pharmacol. actions include
anti-inflammation. With respect to inflammation, in vitro, it
inhibits the activation of **free radical**
-activated transcription factors, such as nuclear factor .kappa.B (
NF.kappa.B) and AP-1, and reduces the prodn.
of pro-inflammatory cytokines such as tumor necrosis factor-.alpha.
(TNF.alpha.), interleukin-1.beta. (IL-1.beta.), and interleukin-8.
Inducible nitric oxide synthase (iNOS) is an inflammation-induced enzyme
that catalyzes the prodn. of nitric oxide (NO), a mol. that may lead to
carcinogenesis. Here, it is reported that in ex vivo cultured BALB/c

mouse

peritoneal macrophages, 1-20 .mu.M of curcumin reduced the prodn. of iNOS
mRNA in a concn.-dependent manner. Furthermore, the authors demonstrated
that, in vivo, two oral treatments of 0.5 mL of a 10-.mu.M soln. of
curcumin (92 ng/g of body wt.) reduced iNOS mRNA expression in the livers
of lipopolysaccharide(LPS)-injected mice by 50-70%. Although many hold
that curcumin needs to be given at dosages that are unattainable through
diet to produce an in vivo effect, we were able to obtain potency at
nanomoles per g of body wt. This efficacy is assocd. with two
modifications in our prepn. and feeding regimen: 1) an aq. soln. of
curcumin was prepd. by initially dissolving the compd. in 0.5 N NaOH and

then immediately dilg. it in PBS; and 2) mice were fed curcumin at dusk after fasting. **Inhibition** was not obsd. in mice that were fed ad lib., suggesting that food intake may interfere with the absorption of curcumin.

ST curcumin nitric oxide synthase gene anticancer; NO synthase gene expression curcumin carcinogenesis

IT Polyphenols (nonpolymeric)

RL: BAC (Biological activity or effector, except adverse); FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (dietary phytochem; inhibition of nitric oxide synthase gene

expression

by curcumin, a cancer preventive natural product with anti-inflammatory properties)

IT mRNA

RL: BPR (Biological process); BIOL (Biological study); PROC (Process) (for iNOS; inhibition of nitric oxide synthase gene expression by curcumin, a cancer preventive natural product with anti-inflammatory properties)

IT Absorption

Anti-inflammatory drugs

Antitumor agents

Food

Gene expression

Peritoneal macrophage

Transformation (neoplastic)

(inhibition of nitric oxide synthase gene expression by curcumin, a cancer preventive natural product with anti-inflammatory properties)

IT Natural products

RL: BAC (Biological activity or effector, except adverse); FFD (Food or feed use); THU (Therapeutic use); BIOL (Biological study); USES (Uses) (inhibition of nitric oxide synthase gene expression by curcumin, a cancer preventive natural product with anti-inflammatory properties)

IT 458-37-7, Curcumin

RL: BAC (Biological activity or effector, except adverse); BPR

(Biological

process); THU (Therapeutic use); BIOL (Biological study); PROC (Process); USES (Uses)

(inhibition of nitric oxide synthase gene expression by curcumin, a cancer preventive natural product with anti-inflammatory properties)

IT 10102-43-9, Nitric oxide, biological studies 125978-95-2, Nitric oxide synthase

RL: BPR (Biological process); BIOL (Biological study); PROC (Process) (inhibition of nitric oxide synthase gene expression by curcumin, a cancer preventive natural product with anti-inflammatory properties)

L6 ANSWER 3 OF 4 BIOSIS COPYRIGHT 2000 BIOSIS

DUPLICATE 1

AN 1996:465207 BIOSIS

DN PREV199699187563

TI Redox regulation of transcriptional activators.

AU Sun, Yi (1); Oberley, Larry W.

CS (1) Parke-Davis Pharm. Res., Div. Warner-Lambert Co., 2800 Plymouth Rd., Ann Arbor, MI 48105 USA

SO Free Radical Biology & Medicine, (1996) Vol. 21, No. 3, pp. 335-348. ISSN: 0891-5849.

DT General Review

LA English

AB Transcription factors/activators are a group of proteins that bind to specific consensus sequences (cis elements) in the promoter regions of downstream target/effector genes and transactivate or repress effector gene expression. The up- or downregulation of effector genes will ultimately lead to many biological changes such as proliferation, growth suppression, differentiation, or senescence. Transcription factors are subject to transcriptional and posttranslational regulation. This review will focus on the redox (reduction/oxidation) regulation of

transcription factors/activators with emphasis on p53, AP-1, and **NF-kappa-B**. The ~~redox~~ regulation of transcriptional activators occurs through highly conserved cysteine residues in the DNA binding domains of these proteins. In vitro studies have shown that reducing environments increase, while oxidizing conditions inhibit sequence-specific DNA binding of these transcriptional activators. When intact cells have been used for study, a more complex regulation has been observed. Reduction/oxidation can either up- or downregulate DNA binding and/or transactivation activities in transcriptional activator-dependent as well as cell type-dependent manners. In general, reductants decrease p53 and **NF-kappa-B** activities but dramatically activate AP-1 activity. Oxidants, on the other hand, greatly activate **NF-kappa-B** activity. Furthermore, redox-induced biochemical alterations sometimes lead to change in the biological functions of these proteins. Therefore, differential regulation of these transcriptional activators, which in turn, regulate many target/effector genes, may provide an additional mechanism by which small antioxidant molecules play protective roles in anticancer and antiaging processes. Better understanding of the mechanism of redox regulation, particularly in vivo, will have an important impact on drug discovery for chemoprevention and therapy of human diseases such as **cancer**.

CC Cytology and Cytochemistry - Human *02508
Genetics and Cytogenetics - Human *03508
Biochemical Studies - Nucleic Acids, Purines and Pyrimidines 10062
Replication, Transcription, Translation *10300
Biophysics - Molecular Properties and Macromolecules *10506
Endocrine System - General *17002
Neoplasms and Neoplastic Agents - Pathology; Clinical Aspects; Systemic Effects *24004

BC Hominidae *86215

IT Major Concepts
Biochemistry and Molecular Biophysics; Cell Biology; Endocrine System (Chemical Coordination and Homeostasis); Genetics; Molecular Genetics (Biochemistry and Molecular Biophysics); Oncology (Human Medicine, Medical Sciences)

IT Miscellaneous Descriptors
CANCER; CHEMOPREVENTION; DNA BINDING; NUCLEAR FACTOR KAPPA-B

ORGN Super Taxa
Hominidae: Primates, Mammalia, Vertebrata, Chordata, Animalia

ORGN Organism Name
human (Hominidae)

ORGN Organism Superterms
animals; chordates; humans; mammals; primates; vertebrates

L6 ANSWER 4 OF 4 HCAPLUS COPYRIGHT 2000 ACS
AN 1995:677972 HCAPLUS
DN 123:101823
TI Oxidative stress, HIV and aids: The basis for antioxidant-oriented antiretroviral nucleoside analogs
AU Abou-Shaaban, Rafiq R. A.
CS College Pharmacy, King Saud University, Riyadh, 11451, Saudi Arabia
SO Saudi Pharm. J. (1995), 3(1-2), 1-22
CODEN: SPJOEM; ISSN: 1319-0164
DT Journal; General Review
LA English
CC 1-0 (Pharmacology)
Section cross-reference(s): 14, 15
AB A review, with 186 refs. The HIV seropos. patients are under systemic and intracellular oxidative stress as a result of an excessive prodn. of reactive oxygen species (ROS) combined with the deficiencies of endogenous antioxidants such as glutathione (GSH), cysteine, vitamin E, carotenoids,

zinc-, manganese-contg. superoxide dismutase (Mn-SOD), selenium-contg. GSH peroxidase and catalase in the T-cell subsets. The different sources of ROS in AIDS patients are the activated leukocytes, cytokines and drugs required to control HIV progression, assocd. infections and **cancers**. Several reports suggest the involvement of ROS activated cytoplasmic factors such as nuclear factor .kappa.B (NF-.kappa.B) and tumor necrosis factor .alpha. (TNF-.alpha.) in the regulation of HIV replication. Since the discovery of retroviral cause for AIDS, a wide variety of agents capable of **inhibiting** different sites of viral life cycle were discovered. These agents were found to possess diverse chem. structures and works on different viral or host targets. The viral targets are either specific enzymes (reverse transcriptase, protease or glucosidase) or viral processes (gene expression, viral binding or viral budding) which interfere with the viral multiplication. The retroviral reverse transcriptase has been a popular target for the design and synthesis of anti-HIV drugs. Recent studies have focused on an intracellular target, the NF-.kappa.B, whose stimulation is related to the lowering of the endogenous antioxidant defense system and stimulation of the HIV expression. In spite of the myriad of known synthetic and/or natural **inhibitors** of HIV over the last decade, the AIDS virus still successfully elude all forms of curative therapy. Replenishing antioxidants will have a preventive role in different stages of AIDS disease, assocd. infections and **cancers**. The beneficial effect of **free radical** scavengers depend on biol. compatibility, the dosage used and the appropriate delivery systems that will allow the scavenger to act at the cellular and tissue sites where the **free radicals** are interfering with the normal function and causing injury. In this report, the author wishes to review the justification for a novel anti-AIDS class "Antioxidant-oriented Antiretroviral Nucleoside Analogs" that might provide curative therapy. These drugs will act by **inhibiting** both the reverse transcriptase viral target and host-mediated stimulation of viral replication. Accordingly, the prospective compds. will block the formation of provirus, extend the latency, after HIV integration into host genome, and **inhibit** viral expression. The required structural specification will be discussed. In addn. the pos. effects of the prospective drugs that might lead to the curative therapy are also outlined.

ST review HIV AIDS antioxidant retrovirus nucleoside
IT Antioxidants
Oxidative stress, biological
Virucides and Virustats
(oxidative stress, HIV and aids and basis for antioxidant-oriented antiretroviral nucleoside analogs)

IT Nucleosides, biological studies
RL: BAC (Biological activity or effector, except adverse); THU
(Therapeutic use); BIOL (Biological study); USES (Uses)
(oxidative stress, HIV and aids and basis for antioxidant-oriented antiretroviral nucleoside analogs)

IT Virus, animal
(human immunodeficiency 1, oxidative stress, HIV and aids and basis for antioxidant-oriented antiretroviral nucleoside analogs)

L8 ANSWER 1 OF 4 HCAPLUS COPYRIGHT 2000 ACS DUPLICATE 1
 AN 1999:100268 HCAPLUS
 DN 130:294707
 TI The nuclear factor- κ B RelA transcription factor is constitutively
 activated in human pancreatic adenocarcinoma cells
 AU Wang, Weixin; Abbruzzese, James L.; Evans, Douglas B.; Larry, Lillie;
 Cleary, Karen R.; Chiao, Paul J.
 CS Departments of Surgical Oncology, The University of Texas M. D. Anderson
 Cancer Center, Houston, TX, 77030, USA
 SO Clin. Cancer Res. (1999), 5(1), 119-127
 CODEN: CCREF4; ISSN: 1078-0432
 PB American Association for Cancer Research
 DT Journal
 LA English
 CC 14-1 (Mammalian Pathological Biochemistry)
 Section cross-reference(s): 3
 AB Pancreatic adenocarcinoma is a leading cause of adult **cancer**
 mortality in the United States. Recent studies have revealed that point
 mutation of the K-ras **oncogene** is a common event in pancreatic
cancer, and oncogenesis mediated by Ras may also involve
 activation of Rel/nuclear factor (NF)- κ B
 transcription factors. Furthermore, the c-rel member of Rel/NF
 - κ B transcription factor family was first
 identified as a cellular homolog of the v-rel **oncogene**,
 suggesting that other members of the Rel/NF- κ B
 family are potentially oncogenes. We therefore investigated the
 possibility that Rel/NF- κ B
 transcription factors are activated in pancreatic **cancer**.
 Immunohistochem. anal., Western blot and Northern blot anal.,
 electrophoretic mobility shift assays, and chloramphenicol
 acetyltransferase assays were performed to det. RelA activity in human
 pancreatic adenocarcinomas and normal tissues and nontumorigenic or
 tumorigenic cell lines. RelA, the p65 subunit of NF- κ B,
 was constitutively activated in .apprx.67% (16
 of 24) of pancreatic adenocarcinomas but not in normal pancreatic
 tissues.
 Constitutive RelA activity was also detected in 9 of 11 human pancreatic
 tumor cell lines but not in nontumorigenic Syrian golden hamster cell
 lines. I. κ B.alpha., a previously identified NF- κ B-
 inducible gene, was overexpressed in human
 pancreatic tumor tissues and cell lines, and RelA activation could be
 inhibited by curcumin and dominant-neg. mutants of I. κ B.alpha., raf,
 and MEKK1. This is the first report demonstrating constitutive
 activation
 of RelA in nonlymphoid human **cancer**. These data are consistent
 with the possibility that RelA is constitutively activated by the
 upstream
 signaling pathway involving Ras and mitogen-activated protein kinases in
 pancreatic tumor cells. Constitutive RelA activity may play a key role
 in
 pancreatic tumorigenesis through activation of its downstream target
 genes.
 ST nucleus factor kappaB RelA subunit activation pancreas adenocarcinoma
 IT Genes (animal)
 RL: BOC (Biological occurrence); BPR (Biological process); BIOL
 (Biological study); OCCU (Occurrence); PROC (Process)
 (I. κ B.alpha.; nuclear factor- κ B RelA transcription factor
 is

constitutively activated in human pancreatic adenocarcinoma cells)

IT NF-.kappa.B
 RL: BAC (Biological activity or effector, except adverse); BOC
 (Biological
 occurrence); BIOL (Biological study); OCCU (Occurrence)
 (RelA subunit; nuclear factor-.kappa.B RelA transcription factor is
 constitutively activated in human pancreatic adenocarcinoma cells)

IT DNA
 RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
 (RelA-DNA binding; nuclear factor-.kappa.B RelA transcription factor
 is
 constitutively activated in human pancreatic adenocarcinoma cells)

IT ras gene (animal)
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (involving in upstream signaling pathway; nuclear factor-.kappa.B RelA
 transcription factor is constitutively activated in human pancreatic
 adenocarcinoma cells)

IT Pancreatic adenocarcinoma
 (nuclear factor-.kappa.B RelA transcription factor is constitutively
 activated in human pancreatic adenocarcinoma cells)

IT Signal transduction (biological)
 (upstream signaling pathway; nuclear factor-.kappa.B RelA
 transcription
 factor is constitutively activated in human pancreatic adenocarcinoma
 cells)

IT 142243-02-5, Mitogen-activated protein kinase
 RL: BSU (Biological study, unclassified); BIOL (Biological study)
 (involving in upstream signaling pathway; nuclear factor-.kappa.B RelA
 transcription factor is constitutively activated in human pancreatic
 adenocarcinoma cells)

RE.CNT 52

RE

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L8 ANSWER 2 OF 4 HCAPLUS COPYRIGHT 2000 ACS DUPLICATE 2
 AN 1997:276623 HCAPLUS
 DN 126:328888
 TI Down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T oncogenes in human colonic Caco-2 cells
 AU Cadoret, Axelle; Bertrand, France; Baron-Delage, Sophie; Levy, Peggy; Courtois, Gilles; Gespach, Christian; Capeau, Jacqueline; Cherqui, Gisele
 CS Laboratoire Biologie Cellulaire, Faculte Medecine Saint-Antoine, INSERM U 402, Paris, 75571, Fr.
 SO Oncogene (1997), 14(13), 1589-1600
 CODEN: ONCNES; ISSN: 0950-9232
 PB Stockton
 DT Journal
 LA English
 CC 14-1 (Mammalian Pathological Biochemistry)
 Section cross-reference(s): 3
 AB The products of ras and src proto-oncogenes are frequently activated in a constitutive state in human colorectal **cancer**. In this study the authors attempted to establish whether the tumorigenic progression induced by oncogenic activation of p21ras or pp60c-src in human colonic cells is assocd. with alterations of the activity and expression of nuclear factor .kappa.B (NF-.kappa.B), a transcription factor suspected to participate in the development of **cancer**. To this end, the authors used Caco-2 cells made highly tumorigenic by transfection with an activated Val-12 human Ha-ras gene or with the polyoma middle T (PyMT) **oncogene**, a constitutive activator of pp60c-src tyrosine kinase activity. Compared with control vector-transfected Caco-2 cells, both **oncogene**-transfected cell lines exhibited: (i) decreased constitutive NF-.kappa.B DNA-binding activity and NF-.kappa.B -mediated reporter gene expression, without alteration of their response to TNF-.alpha. for activation of these parameters; (ii) reduced NF-.kappa.B cytosolic stores along with a decreased p65 expression due, at least in part, to destabilization of p65 mRNA; (iii) a decrease in adhesion to extracellular matrix component-coated substrata which was partially cor. when stimulating NF-.kappa.B transcriptional activity with TNF-.alpha.. These results indicate that the tumorigenic progression induced by oncogenic p21ras or PyMT/pp60c-src in human colonic Caco-2 cells is assocd. with a down-regulation of p65 expression and NF-.kappa.B activity which could be responsible for the reduced adhesive properties of these cells after **oncogene** transfection.
 ST ras src colon cancer NFkappaB expression; T antigen colon cancer NFkappaB expression

IT Cell adhesion
Colon adenocarcinoma
Extracellular matrix
Gene expression
Polyomavirus
Transcription regulation
(down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

IT Middle T antigen
Oncogenes (microbial)
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

IT c-Ha-ras gene (animal)
p21c-Ha-ras protein
RL: ADV (Adverse effect, including toxicity); BOC (Biological occurrence);
BIOL (Biological study); OCCU (Occurrence)
(down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

IT Tumor necrosis factor .alpha.
RL: BAC (Biological activity or effector, except adverse); BIOL (Biological study)
(down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

IT NF-.kappa.B
RL: BOC (Biological occurrence); BPR (Biological process); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

IT mRNA
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

IT Phospholipoproteins
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(pp60c-src; down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

IT 141588-29-6, Protein pp60c-src kinase
RL: ADV (Adverse effect, including toxicity); BIOL (Biological study)
(down-regulation of NF-.kappa.B activity and NF-.kappa.B p65 subunit expression by ras and polyoma middle T (pp60c-src activator) oncogenes in human colonic Caco-2 cells in relation to TNF-.alpha. and extracellular matrix adhesive properties)

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SO Semin. Cancer Biol. (1997), 8(2), 103-111
CODEN: SECBE7; ISSN: 1044-579X

PB Academic

DT Journal; General Review

LA English

CC 14-0 (Mammalian Pathological Biochemistry)

AB A review with 103 refs. Rel/NF-.kappa.B
transcription factors play fundamental roles in the immune system. These
structurally-related proteins share common pathways of activation that
involve their release from inhibitory I.kappa.B factors in response to
stimuli. Accumulating evidence also points to a role for Rel and
I.kappa.B proteins in cellular growth control and oncogenesis. The
rearrangement and amplification of genes encoding Rel/NF-.
kappa.B and I.kappa.B proteins in several human
cancers, together with the acute oncogenicity of the retroviral
v-rel oncogene in birds and mammals, suggests a correlation
between their effects on gene expression and their role in malignancy.
This review focuses on the current status of the assocn. of Rel/NF
-.kappa.B and I.kappa.B proteins with neoplastic cell
transformation in vitro and in vivo.

ST review tumor transformation transcription IkappaB RelNFkappaB

IT Transcription factors
RL: ADV (Adverse effect, including toxicity); BPR (Biological process);
BIOL (Biological study); PROC (Process)
(I.kappa.B; Rel/NF-.kappa.B and I.kappa.B factors in oncogenesis)

IT Transformation (neoplastic)
(Rel/NF-.kappa.B and I.kappa.B factors in oncogenesis)

IT NF-.kappa.B
Transcription factors
RL: ADV (Adverse effect, including toxicity); BPR (Biological process);
BIOL (Biological study); PROC (Process)
(Rel; Rel/NF-.kappa.B and I.kappa.B factors in oncogenesis)

L8 ANSWER 4 OF 4 MEDLINE

AN 92069087 MEDLINE

DN 92069087

TI Molecular mechanisms of transformation by the v-rel oncogene.

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NC CA-22443 (NCI)
CA-07175 (NCI)

SO CRITICAL REVIEWS IN ONCOGENESIS, (1991) 2 (4) 293-309. Ref: 93
Journal code: A1Y. ISSN: 0893-9675.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)

LA English

FS Priority Journals

EM 199203

AB Our knowledge of the molecular mechanisms that underlie the diverse
cellular phenotypes collectively called **cancer** has increased
dramatically over the past 20 years. A significant contribution to our
current understanding of **cancer** has come from research into the
behavior of a unique group of viruses, the acutely transforming
retroviruses. The acutely transforming retroviruses contain one, or
occasionally two, genes that are responsible for the transforming
properties of the viruses. These genes, called retroviral oncogenes, have
been transduced from genes present in the normal cellular genome, called
proto-oncogenes. The proto-oncogenes encode diverse proteins that are
important for the regulation of normal cell growth and differentiation.
One such proto-oncogene, the c-rel proto-oncogene, has
recently been shown to encode a member of the Nuclear Factor-kappa B (

NF-kappa B) transcription factor family. The structural and functional relationship between NF-kappa B and the c-rel protein provides a basis for understanding the molecular mechanism of neoplastic transformation by the v-rel protein.

CT Check Tags: Animal; Human; Support, U.S. Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.

*Cell Transformation, Neoplastic: GE, genetics

*NF-kappa B: GE, genetics

*Oncogenes

*Protein-Tyrosine Kinase: GE, genetics

Proto-Oncogenes

*Retroviridae Proteins, Oncogenic: GE, genetics

Transcription, Genetic

CN EC 2.7.1.112 (Protein-Tyrosine Kinase); 0 (NF-kappa B); 0 (Oncogene Proteins v-rel); 0 (Retroviridae Proteins, Oncogenic)

GEN v-rel